

EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	3955	muscle adj tone	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:13
L2	1486	l1 and (pain)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:12
L3	216	l2 and (neuralgia or neuropathic)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:20
L4	18774	(muscle adj tone or spasm or spasticity)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:14
L5	8995	l4 and pain	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:13
L6	2207	l5 and (neuralgia or neuropathic)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:14
L7	7605	(muscle adj tone or spasticity)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:19
L8	1403	l7 and (neuralgia or neuropathic)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:14
L9	829	l7 and ((neuralgia or neuropathic) near pain)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:14
L10	330	l7 and ((neuralgia or neuropathic) near pain)	USPAT; DERWENT	OR	ON	2007/05/10 15:16
L11	23	l10 and (sodium adj channel)	USPAT; DERWENT	OR	ON	2007/05/10 15:16
L12	423	(muscle adj tone and spasticity)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:34
L13	106	l12 and (neuralgia or neuropathic)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:43
L14	224	tolperisone	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:24
L15	43	l14 and l4	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:31

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L16	2	"200059508"	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:33
L17	4	l12 and retigabine	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:35
L18	309	tolperisone or eperisone or silperisone	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:35
L19	45	l18 and ((muscle adj tone) or spasticity)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:36
L20	745	(lidocaine or riluzole) and ((muscle adj tone) or spasticity)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 16:01
L21	455	l20 and (neuralgia or neuropathic)	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:53
L22	451	l21 and pain	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:43
L23	426	l21 and muscle	US-PGPUB; USPAT; DERWENT	OR	ON	2007/05/10 15:53
L24	154	(lidocaine or riluzole) and ((muscle adj tone) or spasticity)	USPAT; DERWENT	OR	ON	2007/05/10 16:02
L25	139	l24 and muscle	USPAT; DERWENT	OR	ON	2007/05/10 16:02



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Are different pharmacokinetic properties of lidocaine versus propafenone in normal as compared to ischemic myocardium responsible for different electrophysiologic effects which help preventing arrhythmias?

Puddu PE, Rouet R.

PMID: 3167893 [PubMed - indexed for MEDLINE]

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[Myocardial distribution of C-14 lidocaine and C-14 propafenone in relation to regional myocardial blood flow in a model of chronic-phase myocardial infarct] [Cardiologia. 1988]

A comparative evaluation of the effects of propafenone and lidocaine on early ventricular arrhythmias after acute myocardial infarction. [J Int Med. 1988]

Regional myocardial lidocaine concentration determines the antidysrhythmic effect in dogs after coronary artery occlusion. [Circulation. 1985]

Hemodynamic and electrophysiologic effects of combined infusion of lidocaine and propafenone in humans. [J Clin Pharmacol. 1987]

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Links

Oral tocainide versus disopyramide: a double-blind, randomized, crossover study of outpatients with stable ventricular premature beats.

McLaran CJ, Hossack KF, Neilson GH, Siskind V.

Oral tocainide and disopyramide were compared in a double-blind, crossover trial in 10 outpatients with stable ventricular premature beats (VPBs). Efficacy was assessed by suppression of VPB activity and reduction of VPB grade during an exercise test and an 18-h Holter recording. An estimate of variance of VPB activity was obtained from two separate placebo periods, and a 95% confidence limit for VPB suppression was calculated. During Holter recording, both drugs produced a significant reduction in VPB frequency and grade (p less than 0.05). During the exercise challenge, tocainide produced a reduction in VPB frequency and grade, but this did not reach statistical significance; VPB frequency was unaltered by disopyramide, but VPB grade was significantly reduced (p less than 0.01).

PMID: 6206321 [PubMed - indexed for MEDLINE]

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Lorcainide for high-frequency ventricular arrhythmia: preliminary results of a short-term double-blind and placebo-controlled crossover study and long-term follow-up. [Am J Cardiol. 1984]

Prophylaxis against ventricular arrhythmias in suspected acute myocardial infarction: a comparison of tocainide and disopyramide. [Am J Cardiol. 1984]

Comparative study of encainide and disopyramide in chronic ventricular arrhythmias: a double-blind placebo-controlled crossover study. [Am J Cardiol. 1985]

[Comparative studies of tocainide and propafenone in the treatment of ventricular arrhythmias]. [Schweiz Med Wochenschr. 1984]

Tocainide and metoprolol: an efficacious therapeutic combination in the treatment of premature ventricular beats. [Clin Cardiol. 1989]

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Links

[Comparative studies of tocainide and propafenone in the treatment of ventricular arrhythmias]

[Article in German]

Gebhardt A, Schmuderer R, Hilpert P.

In 15 patients with ventricular arrhythmias without previous treatment the effectiveness of 2-amino-2',6'-dimethylpropionanilide (tocainide, Xylotocan) and propafenone was compared in a cross-over-trial. 14 Patients had ventricular arrhythmias of the Lown classification IVa-V, 1 patient of Lown classification III. In 3 patients after the first course of tocainide resp. propafenone no change in therapy was made because of reducing the Lown classification by two or more grades. All patients had 24-h-ECG-Holter-Monitoring before, at the 4th day before change of therapy, and at the 9th day of therapy. Following relevant results were found: Both tocainide (p less than 0,005) and propafenone (p less than 0,05) reduce the total amount of ventricular premature beats (VPB) without statistical significant difference between both substances. Both tocainide (p less than 0,005) and propafenone (p less than 0,02) reduce the number of isolated polytopic VPB without statistical significant difference between both substances. Only tocainide (p less than 0,005) reduces couplets, but there was no statistical significant difference to propafenone. Neither tocainide nor propafenone alone was able to make total suppression of the ventricular arrhythmias in the patients included in this study. Both substances were able to lower VPB both with a more favourable but statistically not significant therapeutic effect of tocainide.

PMID: 6428425 [PubMed - indexed for MEDLINE]

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Canine digitalis arrhythmia as a model for detecting Na-channel blocking antiarrhythmic drugs: a comparative study using other canine arrhythmia models and the new antiarrhythmic drugs, propafenone, tocainide, and SUN 1165. [Heart Vessels. 1985]

Oral tocainide versus disopyramide: a double-blind, randomized, crossover study of outpatients with stable ventricular premature beats. [J Cardiovasc Med. 1994]

[Combination of sotalol with the class I B substances mexiletine or tocainide in complex ventricular extrasystole] [Z Kardiol. 1987]

[Diprafenone--comparative study of anti-arrhythmia therapy with propafenone] [Z Kardiol. 1988]

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Richard E. Klabunde, Ph.D.

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General Pharmacology

Effects on depolarization. Sodium-channel blockers comprise the Class I antiarrhythmic compounds according to the Vaughan-Williams classification scheme. These drugs bind to and block the fast **sodium** channels that are responsible for the rapid depolarization (phase 0) of fast-response cardiac action potentials. This type of action potential is found in non-nodal, cardiomyocytes (e.g., atrial and ventricular myocytes; purkinje tissue). Because the slope of phase 0 depends on the activation of fast **sodium**-channels and the rapid entry of **sodium** ions into the cell (Figure: Na^+ in), blocking these channels decreases the slope of phase 0, which also leads to a decrease in the amplitude of the action potential. In contrast, nodal tissue action potentials (sinoatrial and atrioventricular nodes) do not depend on fast **sodium** channels for depolarization; instead, phase 0 depolarization is carried by calcium currents. Therefore, **sodium-channel** blockers have no direct effect on nodal tissue, at least through the blockade of fast **sodium**-channels.

Fast

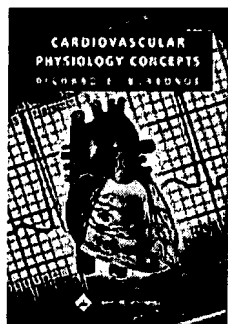
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The principal effect of reducing the rate and magnitude of depolarization by block decrease in conduction velocity in non-nodal tissue (atrial and ventricular muscle). The faster a cell depolarizes, the more rapidly adjacent cells will become depolarized, leading to regeneration and transmission of action potentials between cells. Therefore, block of conduction velocity of action potential transmission within the heart (reduced conduction velocity) can serve as an important mechanism for suppressing tachycardias that are caused by reentry mechanisms). By depressing abnormal conduction, reentry mechanisms

Effects on repolarization. Besides affecting phase 0 of action potentials, sodium-channel blockers may also alter the action potential duration (APD) and effective refractory period (ERP). Because some sodium-channel blockers increase the ERP (Class IA), while others decrease the ERP (Class IB) or have no effect on ERP (Class IC), the Vaughan-Williams classification recognizes these differences as subclasses of Class I antiarrhythmic drugs. These effects on ERP are not directly related to sodium channel blockade, but instead are related to drug actions on potassium channels involved in phase 3 repolarization of action potentials. These channels regulate potassium efflux from the cell (K^+ out), and therefore repolarization. The drugs in these subclasses also differ in their efficacy for reducing the slope of phase 0, with IC drugs having the greatest and IB drugs having the smallest effect on phase 0 (IA drugs are intermediate in their effect on phase 0). The following summarize these differences:

Sodium-channel blockade:
IC > IA > IB

Increasing the ERP:
IA > IC > IB (decreases)

Increasing or decreasing the APD and ERP can either increase or decrease arrhythmogenesis, depending on the underlying cause of the arrhythmia. Increasing APD can interrupt tachycardia caused by reentry mechanisms by prolonging the duration of the action potential (its refractory period). This can prevent reentry currents from re-exciting the tissue. Decreasing the APD can precipitate *torsades de pointes*, a type of ventricular tachycardia caused by reentry mechanisms.

Effects on automaticity. By mechanisms not understood and unrelated to block of sodium channels, antiarrhythmics can suppress abnormal automaticity by decreasing the slope of pacemaker currents.

Indirect vagal effects. The direct effect of Class IA antiarrhythmic drugs on cardiac conduction is modified by their anticholinergic actions. Inhibiting vagal activity can lead to an increase in atrioventricular conduction, which can offset the direct effects of the drugs. For example, if a drug may effectively depress atrial rate during flutter, it can lead to an increase in the number of impulses conducted through the atrioventricular node, necessitating concomitant treatment with a beta-blocker or calcium-channel blocker. These anticholinergic actions are most prominent at the sinoatrial and atrioventricular nodes, which are extensively innervated by vagal efferent nerves. Different drugs within the IA class differ in their anticholinergic actions (see table below).

Specific Drugs and Therapeutic Indications

The following table summarizes Class I compounds in terms of their therapeutic distinguishing characteristics. More detailed information on specific drugs can

Class IA: atrial fibrillation, flutter; supraventricular & ventricular tachy		
quinidine*	anticholinergic (moderate)	cinchonism (blurred psychosis); crampin digitalis toxicity
procainamide	anticholinergic (weak); relatively short half-life	lupus-like syndrome
disopyramide	anticholinergic (strong)	negative inotropic e.
Class IB: ventricular tachyarrhythmias (VT)		
lidocaine*	IV only; VT and PVCs	good efficacy in iscl
tocainide	orally active lidocaine analog	can cause pulmonar
mexiletine	orally active lidocaine analog	good efficacy in iscl
phenytoin	digitalis-induced arrhythmias	
Class IC: life-threatening supraventricular tachyarrhythmias (SVT) and tachyarrhythmias (VT)		
flecainide*	SVT	can induce life-threa
propafenone	SVT & VT;	β -blocking and Ca^{+} can worsen heart fai
morizine	VT; IB activity	

* prototypical drug

Abbreviations: IV, intravenous; PVC, premature ventricular complex.

Side Effects and Contraindications

The anticholinergic effects of IA drugs can produce tachycardia, dry mouth, urinary constipation. Diarrhea, nausea, headache and dizziness are also common side effects. Quinidine enhances digitalis toxicity, especially if hypokalemia is present. Quinidine can precipitate torsades de pointes (especially in patients with long-QT syndrome) caused by afterdepolarizations. Disopyramide is contraindicated for patients with heart failure because of its negative inotropic actions; propafenone can also depress inotropy and increase the increased risk of sudden death in patients with a prior history of myocardial infarction and arrhythmias.

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